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Higher recipient body mass index is associated with post-transplant delayed kidney graft function

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To examine whether a higher body mass index (BMI) in kidney recipients is associated with delayed graft function (DGF), we analyzed data from 11,836 hemodialysis patients in the Scientific Registry of Transplant Recipients who underwent kidney transplantation. The patient cohort included women, blacks, and diabetics; the average age was 49 years; and the mean BMI was 26.8 kg/m^2. After adjusting for relevant covariates, multivariate logistic regression analyses found that one standard deviation increase in pretransplant BMI was associated with a higher risk of DGF (odds ratio (OR) 1.35). Compared with patients with a pretransplant BMI of 22–24.99 kg/m^2, overweight patients (BMI 25–29.99 kg/m^2), mild obesity patients (BMI 30–34.99 kg/m^2), and moderate-to-severe obesity patients (BMI 35 kg/m^2 and over) had a significantly higher risk of DGF, with ORs of 1.30, 1.42, and 2.18, respectively. Similar associations were found in all subgroups of patients. Hence, pretransplant overweight or obesity is associated with an incrementally higher risk of DGF.

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KEYWORDS: body mass index; delayed graft function; kidney transplantation; obesity; pretransplant weight; weight reduction

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Delayed graft function (DGF) is a well-known complication affecting kidney allograft outcomes in the immediate post-transplantation period and is defined as the need for at least one session of dialysis treatment in the first week after receiving a kidney transplant;[1] DGF is attributed to ischemia reperfusion and immunological injury of the graft.[2] The prevalence of DGF varies from 4 to 10% in living donor[2] and 5–50% in deceased donor kidney transplants.[3–7] The occurrence of DGF may significantly complicate the immediate post-transplant management by increasing morbidity and mortality,[8,9] prolonging patient hospitalization,[10] and inflating health care costs.[10–12]

Overweight (body mass index (BMI) 25-$<$30 kg/m^2) and obesity (BMI $>$ 30 kg/m^2) at the time of kidney transplantation are common among North American dialysis patients.[13] Pretransplant obesity may have differential effects on short- versus long-term post-transplant outcomes. Some studies report poorer long-term post-kidney transplant outcomes in obese dialysis patients[14–17] mainly due to cardiovascular complications,[18] whereas other studies have found no association between pretransplant BMI and long-term post-transplant outcomes,[19–22] including our recent study in10,090 kidney transplant recipients.[23] In contrast, pretransplant obesity is usually associated with such untoward short-term complications, such as surgical wound infections or dehiscence.[24] More recent studies report that obese renal transplant recipients have higher risk of developing diabetes mellitus or diverse postoperative complications.[19,22,24–26] However, it is not known whether overweight or obesity has a negative impact on other short-term complications, in particular DGF. To the best of our knowledge, only a small case–control study ($n = 80$) by Espejo *et al.*[27] showed that obese patients have higher risk of DGF after kidney transplantation, whereas Yamamoto *et al.*[28] ($n = 28$) found no meaningful association between obesity and DGF. Obesity is associated with higher sympathetic activity,[29,30] which along with imminent administration of calcineurin inhibitors may lead to renal vasoconstriction and decreased kidney

perfusion, resulting in DGF. Moreover, obesity is associated with longer operative time and longer ischemic time,[31] which is associated with elevated risk of DGF.[32,33] Given these biologically plausible hypotheses and the foregoing inconsistent data, we sought to examine whether recipients' high BMI has a bearing on early post-transplant graft function in a large and contemporary, incident cohort of kidney transplant recipients throughout the United States. We hypothesized that higher pretransplant BMI during the months immediately before kidney transplantation is associated with higher prevalence of DGF in post-transplant patient.

RESULTS

The original 5-year (July 2001–June 2006) national database of all DaVita dialysis patients included 164,789 adult subjects. This database was linked via unique identifiers to the national Scientific Registry of Transplant Recipients (SRTR) registry that included all transplant waitlisted people and kidney transplant recipients until June 2007 (Figure 1). Out of 37,766 DaVita dialysis patients who were identified in the SRTR database, 17,629 had undergone one or more kidney transplantations during their life time, including 14,508 patients who had undergone their first kidney transplantation between July 2001 and July 2007. After excluding those without electronically recorded data ($n=1$), peritoneal dialysis patients ($n=2092$), subjects who lacked data from baseline quarter, or those with outlier values for age (>99 or <16 years; $n=579$), there were 11,836 hemodialysis patients who met all inclusion and exclusion criteria and who subsequently underwent their first kidney transplantation during the observation period.

Table 1 compares the demographic, clinical, transplant-related, and pretransplant laboratory characteristics of the patients with ($n=2628$) and without ($n=9208$) DGF. Patients with DGF were 2 years older and more likely to be diabetic or African American or to have Medicare as their primary insurance. Patients with DGF had lower serum albumin and hemoglobin levels and were more likely to receive kidneys from deceased donors with longer cold ischemic time. Additionally, patients with DGF had a higher pretransplant BMI by 1.2 kg/m^2 than those without DGF (Table 1).

Table 2 shows the results of multivariate logistic regression analyses. Pretransplant BMI was an important predictor of DGF in univariate analysis. One s.d. (s.d. = 6.0 kg/m^2) increase of pretransplant BMI was associated with 30% higher risk of DGF (odds ratio (OR) = 1.30; 95% confidence interval (CI): 1.24–1.36). The association between pretransplant BMI and the risk of DGF in the entire cohort are shown in Figure 2 and Supplementary Figure S1 in the Appendix online. After adjusting for case mix and malnutrition–inflammation complex syndrome variables, pretransplant BMI remained an independent and significant predictor of DGF (Table 2). This association remained significant after adjusting for transplant-related variables: 1 s.d. increase of pretransplant BMI was associated with a 35% higher risk of DGF (OR = 1.35; 95% CI: 1.27–1.45). Compared with patients with pretransplant with BMI in high normal range (22–24.99 kg/m^2), the patient groups with overweight (25–29.99 kg/m^2), mild obesity (30–34.99 kg/m^2), and moderate-to-severe obesity ($\geqslant 35$ kg/m^2) had 30, 42, and 118%, respectively, higher risk of DGF in the fully adjusted model

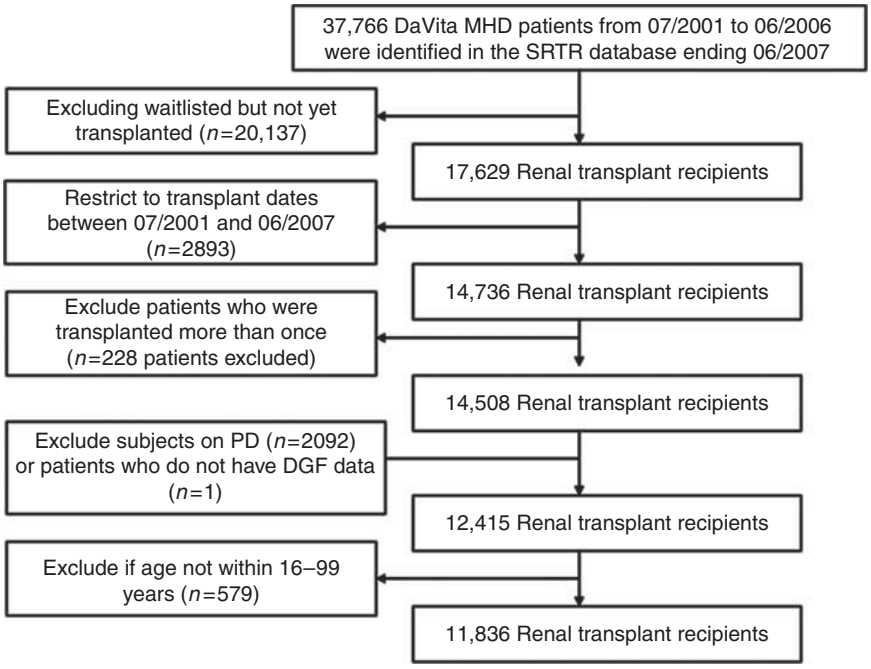

Figure 1 | Flow chart of the patient selection (see text). DGF, delayed graft function; MHD, maintenance hemodialysis; PD, peritoneal dialysis; SRTR, Scientific Registry of Transplant Recipients.

Table 1 | Demographic, clinical, and laboratory characteristics for 11,836 long-term hemodialysis patients who received kidney transplants

Variable	All	With DGF	Without DGF	*P*-value
N (%)	11,836 (100)	2628 (22.2)	9208 (77.8)	NA
Age (years)	49 ± 14	50 ± 13	48 ± 14	<0.001
Gender (% women)	38	34	39	<0.001
Diabetes mellitus (%)	26	29	26	<0.001
Race/ethnicity (%)				
Whites	46	39	48	<0.001
African Americans	27	35	25	<0.001
Hispanics	14	14	14	0.85
Asians	4	3	4	0.01
Dialysis vintage time (%)				
<6 months	12	6	14	<0.001
6–24 months	28	19	31	<0.001
2–5 years	36	41	35	<0.001
>5 years	24	34	21	<0.001
Primary insurance (%)				
Medicare	52	59	50	<0.001
Medicaid	3	3	3	0.47
Private insurance	16	14	17	0.003
Other	20	14	22	<0.001
Marital status (%)				
Married	47	46	48	0.26
Divorced	6	6	6	0.65
Single	27	28	27	0.17
Widowed	3	3	3	0.98
BMI (kg/m^2)	26.8 ± 6.0	28.0 ± 6.7	26.4 ± 5.7	<0.001
Kt/V (dialysis dose)	1.61 ± 0.35	1.60 ± 0.33	1.62 ± 0.36	0.055
nPCR (g/kg/day)	1.05 ± 0.25	1.06 ± 0.25	1.05 ± 0.26	0.01
Serum albumin (g/dl)	4.02 ± 0.37	4.00 ± 0.37	4.03 ± 0.38	<0.001
Creatinine (mg/dl)	10.6 ± 3.2	11.1 ± 3.1	10.5 ± 3.2	<0.001
Bicarbonate (mg/dl)	21.9 ± 3.4	22.2 ± 3.3	21.8 ± 3.4	<0.001
TIBC (mg/dl)	212 ± 40	208 ± 39	213 ± 41	<0.001
Ferritin (ng/ml)[a]	469 (249–731)	534 (299–786)	448 (236–717)	<0.001
Phosphorus (mg/dl)	5.95 ± 1.54	5.97 ± 1.57	5.94 ± 1.53	0.41
Calcium (mg/dl)	9.43 ± 0.74	9.42 ± 0.77	9.44 ± 0.73	0.23
Blood hemoglobin (g/dl)	12.3 ± 1.2	12.2 ± 1.3	12.3 ± 1.2	0.001
WBC (× 10^3/l)	6.8 ± 2.0	6.9 ± 2.1	6.8 ± 2.1	0.24
Lymphocyte (% total of WBC)	23 ± 8	23 ± 8	23 ± 8	0.22
Pretransplant transfusion (%)	31	36	30	<0.001
Number of HLA mismatches	4 (3–5)	4 (2–5)	4 (3–5)	<0.001
PRA (%)[a]	0 (0–3)	0 (0–4)	0 (0–3)	0.21
Cold ischemia time (hours)[a]	14 (4–22)	19 (12–25)	12 (2–20)	<0.001
EDC kidney (%)	19	23	17	<0.001
Donor type (% of living)	32	10	38	<0.001
Donor age (years)	39 ± 15	42 ± 15	38 ± 15	<0.001

Abbreviations: BMI, body mass index; DGF, delayed graft function; EDC, extended donor criteria; HLA, human leukocyte antigen; IQR, interquartile range; NA, not available; nPNA, normalized protein nitrogen appearance; PRA, panel reactive antibody (last value before transplant); TIBC, total iron-binding capacity; WBC, white blood cell.
[a]Median (IQR).
Data are from the last or second-to-last calendar quarter before transplantation. Values are in percentage or mean ± s.d. or median (IQR), as appropriate.

($P<0.05$; Figure 2). Patients with pretransplant BMI higher than 35 kg/m^2 had 87% higher risks of DGF than individuals with pretransplant BMI lower than 35 kg/m^2 (OR = 1.87; 95% CI: 1.52–2.30). Qualitative similar results were found when different cutoff points for BMI were used (Table 2). The association of BMI with DGF was monotonously incremental when BMI was modeled as a continuous variable and using fractional polynomials and cubic splines (Supplementary Figure S1 online). These associations persist in sensitivity analyses including after inclusion of peritoneal dialysis patients (Supplementary Figure S2 online).

Similar associations were observed in all subgroups. Figure 3 shows fully adjusted OR (and 95% CI) of DGF associated with each s.d. higher pretransplant BMI across various patient subgroups. The OR of DGF across all examined subgroups was >1, indicating a higher risk. Most interaction tests did not exhibit small *P*-values, indicating lack of major effect modification by the examined characteristics, except for diabetes and extended donor criteria. The association between pretransplant BMI and DGF was stronger in non-diabetic patients and in recipients of an extended donor criteria kidney (Supplementary Table S1 online). Of note, in deceased donor subgroup, each s.d. increase of BMI was associated with 36% risk of DGF (OR (95% CI): 1.36 (1.26–1.46)). In living donor subgroup, each s.d. increase of BMI was associated with 33% (OR (95% CI): 1.33 (1.14–1.56)) risk of DGF. The interaction term was not significant ($P=0.88$; Supplementary Table S1 online).

DISCUSSION

In 11,836 kidney transplant recipients with comprehensive pre- and post-transplant data, higher pretransplant BMI during the last calendar quarter of hemodialysis treatment was associated with higher risk of DGF during the first post-transplant week. Compared with patients with pretransplant BMI between 22 and 24.99 kg/m^2, the overweight and obese patients with higher pretransplant BMI (25–29.99 kg/m^2, 30–34.99 kg/m^2, and ⩾35 kg/m^2) had incrementally higher risk, that is, 30, 42, and 118% higher risk of DGF, whereas lower BMI <22 kg/m^2 tended to show ~25% lower DGF risk. The associations between pretransplant BMI and DGF were rather consistent across diverse demographic, clinical, and laboratory subgroups. These finding may have important implications for pretransplant management of waitlisted patients.

DGF is a common short-term post-transplant complication and occurs in 5–50% of all kidney transplant recipients. It is especially more frequent with deceased donor kidneys.[3–6] The well-known deleterious effects of DGF in the immediate post-transplant period are multiple and include complications of the immediate post-transplant patient care in the hospital. However, there may be even long-term impact of DGF. Most,[34,35] but not all,[36,37] studies report an association between DGF and reduced long-term graft survival rate. A systematic review reported that DGF is associated with a 41% increased risk of graft loss,[8] 38% increased risk of acute rejection in the first year, and a higher serum creatinine concentration at 3.5 years of follow-up.[8]

Overweight and obesity are highly prevalent at the time of kidney transplantation.[13] Previous reports have described

Table 2 | Multivariate logistic regression models showing pretransplant weight and BMI and their ORs and 95% CI for delayed graft function

	Unadjusted		Case mix adjusted		Case mix and MICS adjusted		Case mix, MICS, and transplant data adjusted	
Pretransplant weight	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Weight (kg; +1 s.d.)	1.29 (1.24–1.35)	<0.001	1.32 (1.25–1.39)	<0.001	1.33 (1.25–1.41)	<0.001	1.34 (1.26–1.44)	<0.001
BMI (kg/m^2; +1 s.d.)	1.30 (1.24–1.36)	<0.001	1.29 (1.23–1.36)	<0.001	1.33 (1.26–1.41)	<0.001	1.35 (1.27–1.44)	<0.001
BMI > 25 (kg/m^2) vs BMI ⩽ 25 (kg/m^2) (ref.)	1.48 (1.44–1.75)	<0.001	1.53 (1.38–1.69)	<0.001	1.53 (1.37–1.72)	<0.001	1.57 (1.39–1.79)	<0.001
BMI > 30 (kg/m^2) vs BMI ⩽ 30 (kg/m^2) (ref.)	1.54 (1.39–1.71)	<0.001	1.51 (1.35–1.68)	<0.001	1.50 (1.33–1.69)	<0.001	1.48 (1.30–1.70)	<0.001
BMI > 35 (kg/m^2) vs BMI ⩽ 35 (kg/m^2) (ref.)	1.78 (1.53–2.08)	<0.001	1.82 (1.55–2.13)	<0.001	1.84 (1.53–2.21)	<0.001	1.87 (1.52–2.30)	<0.001
BMI > 40 (kg/m^2) vs BMI ⩽ 40 (kg/m^2) (ref.)	2.25 (1.72–2.96)	<0.001	2.34 (1.77–3.10)	<0.001	2.51 (1.80–3.50)	<0.001	2.78 (1.88–4.12)	<0.001

Abbreviations: BMI, body mass index; CI, confidence interval; MICS, malnutrition–inflammation complex syndrome; OR, odds ratio.

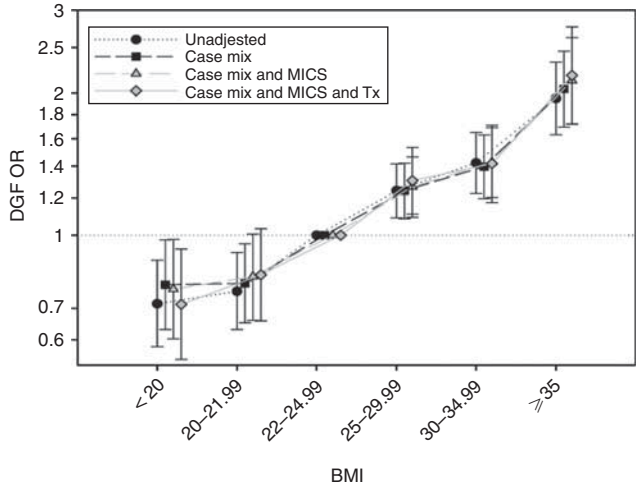

Figure 2 | Multivariate analysis of logistic regression models showing pretransplant body mass index (BMI) and odds ratio (OR, and 95% confidence interval as error bars) of delayed graft function (DGF) in four different models (reference: BMI 22 to <25 kg/m^2). MICS, malnutrition–inflammation complex syndrome; Tx, transplanted covariates.

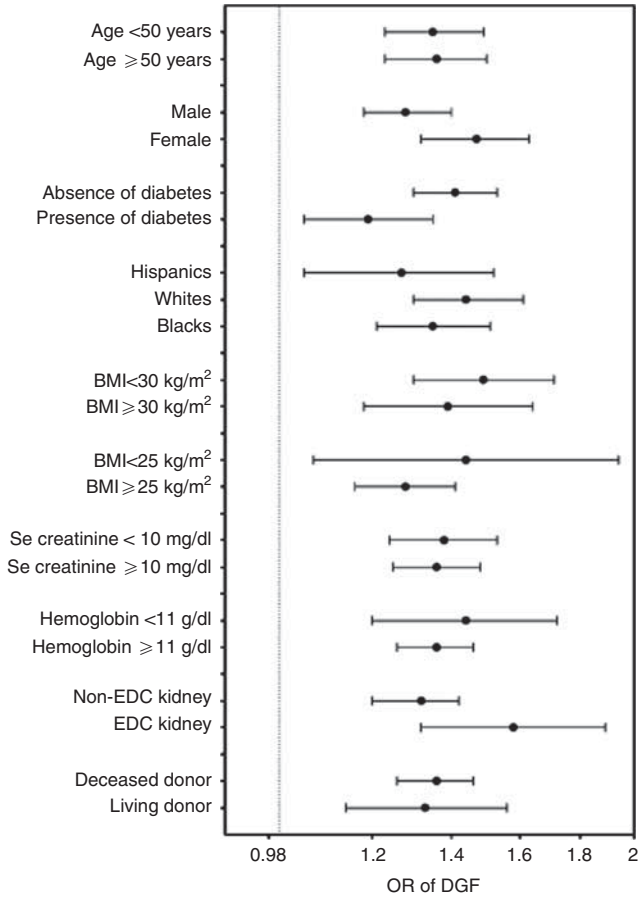

Figure 3 | Multivariate analysis of fully adjusted (for case mix, malnutrition–inflammation complex syndrome, and transplant covariates) logistic regression models showing pretransplant body mass index (BMI) and odds ratio (OR, and 95% confidence interval as error bars) of delayed graft function (DGF) for each standard deviation higher BMI in different subgroups of patients. EDC, extended donor criteria.

conflicting associations between BMI and various outcomes in kidney transplant recipients. Early studies showed higher risk of postoperative complications[31] and early surgical wound infections[24] in obese patients. Lentine *et al.*[18] reported higher incidence of cardiovascular event, including heart failure and atrial fibrillation, and early postoperative complications in obese versus non-obese patients. Several other studies, however, did not find any association between pretransplant BMI and mortality.[19,21,22] Chang *et al.*[38] reported that obesity *per se* was not associated with poorer kidney transplant outcomes, although it was associated with factors that led to poorer graft and patient survival. Indeed, patients with a BMI ⩾30 kg/m^2 receiving single pediatric kidneys had better death-censored graft survival rates when compared with non-obese patients.[39] Zaydfudim *et al.*[40] reported that pretransplant overweight and obese status did not affect physical quality of life after kidney transplantation.

In our study, the association between the pretransplant BMI and the risk of DGF was rather linear, incremental, consistent across virtually subgroups, and robust, even after adjusting for several important confounders. Only few studies examined the association between BMI and DGF, and found conflicting or equivocal results. A small case–control study ($n = 80$) showed the obese patients have higher risk of DGF after kidney transplantation,[27] whereas Yamamoto *et al.*[28] ($n = 28$) found no association between obesity and DGF. These studies were likely underpowered and used inconsistent definitions of DGF.

Several potential mechanisms may contribute to the observed associations. A biologically plausible explanation

is that obesity is associated with longer operative time of longer and warm ischemic time,[31] which are *per se* risk factors of DGF.[32,33] Obesity is associated with high sympathetic activity,[29,30] which results in renal vasoconstriction. Moreover, the prompt administration of calcineurin inhibitors after transplantation, probably in higher doses given to overweight or obesity, may aggravate vasoconstriction and further compromise graft perfusion, increasing the risk of DGF. Another potential explanation is the linkage between obesity and increased prothrombotic activity and endothelial dysfunction.[41] Body fat mass, in particular central obesity, is associated with higher levels of thrombin generation.[42,43] Obesity is also a risk factor for venous thromboembolic disease.[44] Increased prothrombotic activity and endothelial dysfunction may contribute to the risk of graft microthrombosis,[45] which *per se* may have an important role in DGF.[46]

There are potential limitations to our study. Like all observational studies, our study too cannot prove causality. Patients who were excluded from analyses were likely different from the included ones, but their proportion was relatively small. In the SRTR data set, more detailed data about immunosuppression therapy such as calcineurin inhibitor dose or blood level or the induction therapy, which may also have an effect on the risk of DGF, do not exist. Additional limitation is the uncertainty about the use of BMI as a measure of obesity. BMI *per se* may not be an appropriate measure to characterize nutritional status, body composition, obesity, or muscle mass in dialysis patients.[47–52] To better characterize nutritional status, additional parameters such as waist circumference would be needed.[48,50–52] To the best of our knowledge, our study is the first examining the association between pretransplant BMI and immediate post-transplant DGF in such a large and nationally representative patient population. Other strengths of our study include the high number of patients, the multilevel adjustments including for laboratory data, and the contemporary nature of the cohort (2001–2007).

CONCLUSIONS

In our large and contemporary national cohort of 11,836 kidney transplant recipients, pretransplant BMI is associated with risk of DGF, even after extensive multivariate adjustment. The association between pretransplant BMI and DGF was consistent in all examined subgroups. Despite data indicating an obesity paradox with greater survival of overweight and obese hemodialysis patients,[47,49,53,54] careful trials of closely supervised weight reduction may be needed to examine whether immediate post-transplant outcomes including risk of DGF can be improved.

MATERIALS AND METHODS

Patients

We linked data on all kidney transplant recipients listed in the SRTR up until June 2007 to a list of individuals with chronic kidney disease stage 5D, who underwent maintenance hemodialysis treatment from July 2001 to June 2006 in one of the outpatient dialysis facilities of a US-based large dialysis organization (DaVita Inc, before its acquisition of former Gambro dialysis facilities). The study was approved by the institutional review committees of both Los Angeles Biomedical Research Institute at Harbor-UCLA and DaVita Clinical Research. The study was conforming to the principles of the Declaration of Helsinki. Because of the large sample size, the anonymity of the patients studied and the non-intrusive nature of the research, the requirement for informed consent was waived.

Clinical and demographic measures

The creation of the national DaVita maintenance hemodialysis patient cohort has been described previously.[51,54–57] Demographic data and details of medical history were collected, including information on age, gender, race, type of insurance, marital status, presence of diabetes, height, post-hemodialysis dry weight (to calculate average BMI), and dialysis vintage. Dialysis vintage was defined as the duration of time between the first day of dialysis treatment and the day of kidney transplantation.

To minimize measurement variability, all repeated measures for each patient during any given calendar quarter, that is, over a 13-week or 3-month interval, up to the time of kidney transplantation, were averaged and the quarterly means in each of the 20 calendar quarters were used in our analyses. Each patient had up to 39 recoded post-hemodialysis weights, corresponding thrice weekly maintenance hemodialysis treatment. All values were averaged into one single quarterly value per patient per each calendar quarter. In the present study, we used the average of a number of BMI measurement in the last quarter before transplantation.

After deleting extreme outliers (BMI <12 or $>60\,kg/m^2$), we divided pretransplant BMI into six *a priori* selected categories or underweight ($\leqslant 19.99\,kg/m^2$), low normal weight (20–21.99 kg/m^2), high normal weight (22–24.99 kg/m^2) overweight (25–29.99 kg/m^2), mild obesity (30–34.99 kg/m^2), and moderate-to-sever obesity ($\geqslant 35\,kg/m^2$). These increments were consistent with our previous studies.[58]

Laboratory measures

Blood samples were drawn using uniform techniques in all of the DaVita dialysis clinics and were transported to the DaVita Laboratory in Deland, Florida, typically within 24 h. All laboratory values were measured by automated and standardized methods in the DaVita Laboratory. Most laboratory values were measured monthly, including serum urea, creatinine, albumin, calcium, phosphorus, bicarbonate, and total iron-binding capacity. Serum ferritin was measured at least quarterly. Hemoglobin was measured, at least, monthly in essentially all patients and weekly to biweekly in most patients. Most blood samples were collected before dialysis, with the exception of the postdialysis serum urea nitrogen that was obtained to calculate urea kinetics. Kt/V (single pool) was calculated using urea kinetic modeling equations, as described elsewhere.[56] Albumin-corrected calcium was calculated by subtracting 0.8 mg/dl for each g/dl serum albumin below 4.0 g/dl.[59]

Definition of DGF

DGF was defined as the need for any dialysis therapy in the first week after transplantation.[1]

Statistical methods

Data were summarized using proportions, means ($\pm$ s.d.), or medians (interquartile range), as appropriate. Categorical variables

were analyzed with χ^2-tests, and continuous variables were compared using Student's *t*-tests or Mann–Whitney U-tests, Kruskal–Wallis H tests or analysis of variance, as appropriate. In all statistics, two-sided tests were used and the results were considered statistically significant if *P*-value was <0.05. Logistic regression models were employed to estimate the OR (and 95% CI) of post-transplant DGF based on pretransplant BMI during the calendar quarter preceding the kidney transplantation.

For each analysis, four models were examined based on the level of multivariate adjustment: (I) an unadjusted model; (II) case mix adjusted models included age, gender, race ethnicity (African Americans and other self-categorized blacks, non-Hispanic whites, Asians, Hispanics, and others), diabetes mellitus, dialysis vintage, primary insurance (Medicare, Medicaid, private, and others), marital status (married, single, divorced, widowed, and other or unknown), the standardized mortality ratio of the dialysis clinic during entry quarter, dialysis dose as indicated by Kt/V (single pool), and presence or absence of a dialysis catheter; (III) malnutrition–inflammation complex syndrome-adjusted models, which included all of the covariates in the case mix model as well as 11 surrogates of nutritional status and inflammation, including 10 laboratory variables with known association with clinical outcomes in HD patients, that is, normalized protein catabolic rate as an indicator of daily protein intake, also known as the normalized protein nitrogen appearance,[60] serum or blood concentrations of albumin, creatinine, total iron-binding capacity, ferritin, phosphorus, calcium, bicarbonate, peripheral white blood cell count, lymphocyte percentage, and hemoglobin; and (IV) case mix, malnutrition–inflammation complex syndrome, and transplant data-adjusted models included all of the above plus seven transplant-related variables: (1) donor type (deceased or living), (2) donor age, (3) panel reactive antibody titer (last value before transplant), (4) number of human leukocyte antigen mismatches, (5) cold ischemia time, (6) transfusion before transplantation, and (7) extended donor criteria using standard definition (donor history of hypertension and/or serum creatinine of donor >1.5 mg/dl and/or cause of death in donor is a cerebrovascular event).

In sensitivity analyses, we reexamined all associations after 1962 peritoneal dialysis patients were added to 11,836 hemodialysis patients, leading to a total sample size of 13,798 kidney-transplanted recipients. Missing covariate data in the last (pretransplant) calendar quarter were imputed by medians or means including from prior calendar quarters, as appropriate. All analyses were carried out using STATA version 11.1 (STATA Corporation, College Station, TX).

DISCLOSURE

MK is an employee of DaVita. KKZ is the medical director of DaVita Harbor-UCLA/MFI in Long Beach, CA. The other authors declared no competing interests.

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SUPPLEMENTARY MATERIAL

Table S1. Multivariate analysis of fully adjusted (for case mix, malnutrition–inflammation complex syndrome, and transplant covariates) logistic regression models showing pretransplant body mass index (BMI) and odds ratio (OR, and 95% confidence interval (CI) as error bars) of delayed graft function (DGF) for each standard deviation higher BMI in different subgroups of patients.

Figure S1. Multivariate analysis of logistic regression models showing pretransplant BMI and OR (and 95% CI as error bars) of DGF in our fully adjusted models using cubic spline curve in 11,836 kidney-transplanted patients.

Figure S2. Multivariate analysis of logistic regression models showing pretransplant BMI and OR (and 95% CI as error bars) of DGF in four different models (reference: BMI 22 to <25 kg/m^2; A) and the fully adjusted model using cubic spline curve (B) in 13,798 kidney-transplanted patients.

Supplementary material is linked to the online version of the paper at http://www.nature.com/ki

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